AN EVALUATION OF PRESENT DAY SURGERY FOR MITRAL STENOSIS*

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PRESENT day surgery for mitral stenosis at the Episcopal Hospital in Philadelphia was begun in 1948.[1] On first thought, it would appear that sufficient time has now elapsed to permit valid evaluation of this type of therapy. There are, however, many reasons why even seven years is too short a time to assess with finality the beneficial effects of mitral valvotomy in individuals with mitral stenosis. Statistically, it appears idle to attempt even to discuss long term beneficial effects because, compared to the natural history of mitral stenosis, which often outlasts the professional life of a physician, the postoperative intervals are far too short to make such a discussion meaningful.

Even short term beneficial effects of surgery are difficult to evaluate because many of the early reports and, indeed, the vast majority of those today, predicate beneficial effects on improvement in functional capacity of individuals after surgery. No one, of course, questions the importance of functional improvement. Indeed, the major function of a physician treating an ill patient is to restore him as close to normal activity as possible. Without considering potential bias of both physician and patient in interpretation of improvement, reports of this sort imply that individuals with mitral stenosis cannot improve unless mitral valvotomy is done. Yet, every physician knows that rheumatic heart disease with mitral stenosis is a chronic illness that may remain stabilized for years and that this stable state may be upset by events producing symptoms and signs, none of which is necessarily specific for mitral stenosis or its degree, and, indeed, some of which may not even be related in any way to the mitral valve. These exacerbations may be unpredictable in time, duration or frequency. Their causes may be manifold and, in themselves, have variable prognoses. Individuals may die in an exacerbation. More often, depending upon

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their causes and severities, the individual's resistance and his cardiac reserve, disappearance of the phenomena characteristic of the exacerbation may occur either with no detectable change in residual cardiac disease or with an increase, or, indeed, at times, with a decrease in recognizable deviations of the heart from normal. In other words, the functional capacity of the individual may change naturally or following medical treatment; and, this change may be recorded in any type of classification adopted.

At the beginning of the present surgical era, it was to be expected that individuals with the most rapid impairment of cardiac function should most eagerly seek a restoration of health by means of this new technique. Surgeons and cardiologists, buttressed by the irresistible logic of the procedure, were reluctant to refuse such individuals surgery. Physicians were told that it was their responsibility to have individuals with mitral stenosis referred to surgeons. The minds of these sick people were properly prepared by ubiquitous and repetitious propaganda in the lay press. The propagandist had already taught us that individuals rarely can resist repetition. Remissions occurring naturally or induced by medical therapy were regarded as indications for surgery rather than evidence that the contracted mitral orifice was not the immediate cause for the change in cardiac function. The implication was that, regardless of the immediate cause for change in functional capacity of the individual, it was the contracted mitral orifice that made the illness serious. Therefore, adequate surgery would prevent such a train of events in the future.

Although there are considerable physiologic data to support such an hypothesis, as there are pathologic data to indicate that surgery is sometimes inadequate, and at other times ineffective even though adequate, it should have been obvious to all that statements of improvement under such circumstances are not justified unless the basis for comparison is the effect of similar precipitating factors in the future or the functional status of the individual before the onset of his exacerbation. No attempt was made to distinguish acute events that tend more frequently to occur early in the life history of mitral stenosis and are frequently self-limited from those that occur late. On the contrary, individuals were and frequently still are lumped together into specific functional classes regardless of age, of cause and duration of disabilities and their response to non-surgical therapy, of heart size,

of the type of heart failure and of the number and frequency of previous exacerbations. Yet, we all know that each one of these factors is important in evaluating prognosis of both the acute exacerbation and the long term course of the illness. It is because of this tendency to equate individuals with similar functional incapacities regardless of cause, duration or the extent of cardiac disease, that we have been unable to accept as valid most of the reports of the percentages and degrees of even short term improvements attributed to surgery. This statement is not to be interpreted as meaning that we believe that mitral valvotomy has no value.

The problems of the cyclic nature of functional changes in individuals with mitral stenosis are part of the larger subject of the natural history of mitral stenosis as affected by the best available medical therapy. Unfortunately, all studies on this subject are as yet incomplete or inconclusive. As a base line, we may use May Wilson's prediction based on studies before the widespread use of antibiotic therapy.[2] Dr. Wilson stated that the survival rate for individuals with rheumatic heart disease twenty years after onset of their disease was 74.9 per cent. She predicted that 50 per cent would live more than 45 years after the initial occurrence of the disease. United States Government statistics tend to support this statement and indicate that the average age at death for all individuals with rheumatic heart disease in 1951 was fifty-five years.[3] According to Wilson, the over-all death rate was 14.7 per thousand per year and the death rate five to ten years after onset was usually less than 1 per cent per year. This mortality can be compared to that of mitral valvotomy that has a minimal mortality of 4 to 5 per cent even for the individual with asymptomatic mitral stenosis. If Wilson's statistical conclusions are correct, there can be no excuse at this time for subjecting asymptomatic individuals with mitral stenosis to operation.

Actually, prognosis of rheumatic individuals has steadily improved during the past century.[4] It has improved rapidly during the time interval of Wilson's study and improvement has been accelerated by the introduction of antibiotic therapy. Furthermore, the age at death is higher for any succeeding age group than any preceding one or than the group as a whole. Thus, at age thirty-seven years, the average age at operation, Wilson's prediction implies that not 50 per cent but 83.4 per cent will survive the age of fifty. Only 16.6 per cent will

have died below the age of fifty. Strangely, this percentage, 16.6 per cent, represents our surgical group mortality within three years of operation of the first 500 individuals subjected to mitral valvotomy.[5] The average age at death was forty years. If we are to improve on Wilson's statistics, it will be necessary for us to have no more deaths in this group for considerably more than ten years. Unfortunately, deaths have occurred at such a rate after this three year study that we are in a situation similar to Tristram Shandy who found himself each succeeding year, 364 days behind the preceding one.

Such a simplification of statistics is unfair in some ways to medicine and in others to surgery. Obviously, the surgical group is composed almost entirely of individuals with symptoms and the medical group may contain a significant percentage of asymptomatic individuals. On the other hand, Wilson's group includes all individuals with rheumatic heart disease and not just those with isolated or predominant mitral stenosis and no distinction is made of the disability causing death. Clearly, not all disabilities and deaths in individuals with mitral stenosis are potentially preventable by mitral valvotomy.

In order to obtain a more distinct picture of the present day problem of mitral stenosis, we have analyzed all deaths of individuals with rheumatic heart disease from 1945, when antibiotic therapy was first widely used, to the birth of present day surgery for mitral stenosis in our hospitals. The details of this study are published elsewhere.[6]

In essence, there were 141 individuals with rheumatic heart disease who died. This group represents 8.2 per cent of 1718 individuals admitted with a clinical diagnosis of rheumatic heart disease which, in turn, represents 0.9 per cent of 188,066, the total hospital admission.

Deaths were divided into seven clinical categories:

1. Sixteen (11.3 per cent) died of diseases unrelated to rheumatic heart disease or its sequelae.

2. Nineteen (13.5 per cent) died of rheumatic carditis. The average age at death was 20.9 years. These individuals all had florid carditis, the type that even the surgical enthusiast refuses to accept for surgery. Moreover, only two had mitral stenosis and one of these had extensive rheumatic pneumonitis. We have followed one such individual subjected to mitral valvotomy. Although surgery was adequate as shown at autopsy, progressive deterioration of the heart and its function occurred until death fifteen months later at the age of

twenty-one. Yet, until the last few days of her life, the patient, her physician and her surgeon were confident that surgery had been effective.

3. Sixteen (11.3 per cent) died of bacterial endocarditis at an average age of 37.4 years. Only one individual had isolated mitral stenosis. He was fifty-six years old with hypertension and a precordial systolic murmur. He was thought to have aortic stenosis but autopsy showed mitral stenosis with an orifice of 1½ fingers. The left ventricle was 1.75 centimeters thick. There were vegetations on the mitral valve and an embolus in the pulmonary artery.

4. Nine (6.4 per cent) died of pulmonary infarction with heart failure at an average age of 41.1 years. Only one had isolated mitral stenosis, the mitral orifice being rigid and 4 centimeters in circumference. He had thrombosis of the right pulmonary artery with infarction of the right middle and lower lobes of the lung.

5. Thirteen (9.2 per cent) died of systemic embolism with preexisting heart failure at an average age of 47.7 years. Only one had isolated mitral stenosis, a fifty-three year old man admitted into the hospital in coma with hypertension and atrial fibrillation. At autopsy, the mitral orifice was 6 centimeters in circumference.

6. Twelve (8.5 per cent) died of systemic embolism without heart failure at an average age of 46.5 years. All had atrial fibrillation. Of the three with autopsies, all had isolated mitral stenosis.

Here then is the first group in which mitral valvotomy, on theoretical grounds, may do good. The practical difficulty lies in predicting which individuals with mitral stenosis and atrial fibrillation are doomed to die of embolism, or, indeed, sustain any type of embolus. Only 25 per cent of those dying of embolism had sustained a preceding one, the interval varying from one to three years. Furthermore, of those sustaining a major embolus with survival, less than one-half will die of a succeeding embolus.[7] The greatest difficulty, however, is that mitral valvotomy is accompanied by the highest percentage of embolism. The incidence is significantly higher for those with atrial fibrillation, varying in different series from 5 to over 10 per cent. Indeed, it is our commonest cause of operative death. It probably occurs slightly more frequently in those with a history of embolism in the past. The incidence of embolism occurring naturally is about 1 per cent per year.[8] Therefore, there can be no excuse for performing

mitral valvotomy as a prophylactic procedure against embolism. Furthermore, it is commonly stated that once an individual passes the hurdle of operation without embolism, there is very little chance of sustaining one in the future. This statement overlooks the fact that the incidence of embolism at operation is so high that it will take from five to twelve years free of embolism to reduce the incidence of surgically produced embolism to that occurring naturally. It also overlooks the fact that most operations are done long before the age period when the incidence of naturally occurring embolism is at its peak.[7] Lastly, in our experience, the statement itself is not true. We recognize embolism long after operation. The incidence is as yet below 1 per cent per year and is higher in those with atrial fibrillation. Our last case developed a fatal cerebral embolus three years after operation during a bout of atrial fibrillation that was not present previously.

Actually, the cardiac surgeon avoids thrombi that are adherent to the auricular wall for fear of producing embolism and thereby permits the threat of future spontaneous embolism to persist.

7. Fifty-six (39.7 per cent) died of congestive heart failure at an average age of 50.9 years. Of twenty autopsies in this group, only four had isolated mitral valve disease and only two of these were reported as stenotic by the pathologist. Only one individual died below the age of fifty years with isolated mitral stenosis and congestive heart failure, a woman of thirty-three years with advanced scleroderma.

From this analysis, it appears that isolated mitral stenosis is a rare cause of fatal congestive heart failure and when it does occur, tends to occur most commonly in the sixth decade. Furthermore, such deaths occur only in individuals with very large hearts and atrial fibrillation. All surgical deaths that occur in individuals without intractable right heart failure are attributable directly to surgery and the age of death can be used to gauge the probable loss of years of life. The average weight of the heart at death in this group was 643 grams. The heart weight in individuals who die after mitral valvotomy may also be used to determine whether premature death has occurred.

It is, however, true that most individuals who die of congestive heart failure have had symptoms for many years and, in our experience, generally for over a decade. The symptoms, their intensity and the incapacity of the patient may vary greatly, little, or not at all from time to time. Mitral valvotomy is based upon the concept that relief

of the impediment to blood flow through the mitral valve will relieve symptoms and incapacity. Unfortunately, the type of incapacity produced by mitral stenosis may be indistinguishable from that produced by mitral stenosis with significant mitral regurgitation and, indeed, by myocardial disease independent of the mitral stenosis that is present. For these reasons, we must not overlook the fact that there may be significant cost of operation due to error in diagnosis. The error even for the diagnosis of the predominant mitral valve pathology cannot, at the present time, be assessed because there is, as yet, no reliable technique for confirming, during life, the accuracy of the surgeon's interpretation of the tactile and kinesthetic sensations of his finger.

The major difficulty in evaluating mitral valvotomy is that it does not destroy the conventionally accepted findings of mitral stenosis. Our experience indicates that the clinical, electrocardiographic and roentgenologic findings in mitral stenosis are so rarely changed after operation, that, when changes are seen, we always search carefully for a transient event in the preoperative past to account for it. Most commonly, these are acute carditis in the younger individual and pulmonary infarction or infection in the older person. We have seen pulmonary infarction at operation totally unsuspected clinically or radiologically.

Indeed, it is more common for additional signs of mitral valve disease to appear after operation, the commonest of which is a systolic murmur or its increase. Most physicians regard this murmur as insignificant. If conventional criteria are used, a diagnosis of mitral regurgitatation must be made. It is, of course, possible that surgical tears of the mitral leaflets produce systolic murmurs of no dynamic significance. However, we believe that mitral regurgitation is actually the cause for most of the murmurs, first, because there is no question of symptom-producing and even fatal mitral regurgitation, so that it is inconceivable that lesser grades of regurgitation are not produced; secondly, even though cardiac dynamics are considerably depressed during operation, regurgitant jets are produced or increased by the surgeon in at least 10 per cent of his operations; thirdly, giant P waves occasionally are seen temporarily immediately after surgery; fourthly, a notched widened P wave may first appear after surgery and persist; and, lastly, these individuals develop after surgery an apex impulse that is localized, forceful and visible, with a distinct thrust, a finding that, in the absence

of chest deformity, has always meant to us an increase in size of the left ventricle. These individuals may rarely show a decrease in the electrocardiographic evidence of right ventricular hypertrophy but this change is always accompanied by obviously increased and high R waves in the left precordial leads. Decrease in rightward deviation of the QRS vector may also occur in individuals who had sustained pulmonary infarction or pneumonia with or without pulmonary edema in the recent preoperative past or in those whose preoperative electrocardiogram was taken before complete dissipation of heart failure.

Because the conventional findings of mitral stenosis are not abolished by surgery, there has been a tendency to evaluate adequacy of operation on the basis of relief of symptoms or incapacity, a method that obviously begs the question of the significance of mitral stenosis and of the adequacy of restoration of mitral valve function and its value.

This fallacy is clearly demonstrated by the surgical production of permanent atrial fibrillation that occurs in approximately 10 per cent of individuals with preoperative sinus rhythm. It has been universally accepted that naturally occurring atrial fibrillation in individuals with rheumatic heart disease represents a more advanced stage of the disease than those with sinus rhythm and that those with atrial fibrillation are more likely to develop embolism and perhaps other complications. Yet, everyone includes individuals with surgically produced atrial fibrillation in the improved group, even though digitalis which had not been required before operation is now necessary. It may be that the surgical production of atrial fibrillation has a different prognosis than that occurring naturally. Certainly, though, it is too early to regard such individuals as improved regardless of how they feel. We believe that, regardless of so-called functional classification, no one who requires more cardiac therapy after operation should, as yet, be included in the group improved by surgery.

The importance of medical therapy can be clearly demonstrated by withholding it. Incidentally, many unoperated individuals with mitral stenosis and sinus rhythm have been unnecessarily maintained on cardiac therapy. Even individuals with atrial fibrillation may remain clinically symptom-free for years without digitalis. Apparently, troublesome fast ventricular rates are prevented by partial organic or functional block at the atrial-ventricular node. Such long continued cardiac therapy that had no relationship to the disappearance of pre-existing

symptoms has greatly complicated the evaluation of additional therapy, medical or surgical.

Another distressingly frequent effect of mitral valvotomy is the appearance, after a variable latent period, of a combination of events that in our experience does not occur in any other type of non-rheumatic cardiac or pulmonary surgery. The syndrome, characterized by a single bout or, more commonly, by recurrent bouts of pain and fever, with a variable incidence of pericarditis, transient atrial fibrillation or atrial tachycardia, prolonged atrial-ventricular conduction time, cardiac enlargement and cardiac failure, all indicating that the reaction is not limited to the pericardium and pleura, has been regarded by us and others as a reactivation of rheumatic fever.[9, 10] Some prefer the non-committal term, post-commissurotomy febrile syndrome. We have what appears to be indisputable evidence of its rheumatic nature, namely, fresh Aschoff bodies along the lines of incision both on the mitral leaflet and the pericardium. Some agree with our interpretation but regard the reaction as unimportant because they regard it as self-limited and without effect on the outcome of the operation. It is recognized today that spontaneous reactivation of rheumatic fever subsides with frequently no detectable increase in permanent cardiac damage. This recovery does not invalidate the diagnosis of recurrence of rheumatic fever but rather is attributed to resistance of the host, cardiac reserve and medical therapy. It is known that young individuals with active rheumatic carditis may show no progressive increase in heart size, if they remain at bed rest.[11] If the postoperative reaction has been intense enough to produce cardiac failure, it is our experience that the ultimate outlook depends upon the preoperative cardiac reserve. Individuals with preoperative intractable right heart failure remain permanently worse and require an intensification of the cardiac therapeutic regimen. Individuals with right heart failure that had cleared before operation have a 50 per cent chance of remaining in permanent right heart failure or require an intensification of therapy to keep them out of it. Individuals with left heart failure that had cleared before operation usually lose all signs of failure that appear after operation, unless excessive mitral regurgitation is produced. But, we believe that it is, as yet, too early to be certain, even in these instances, that reactivation of rheumatic fever was harmless, because even at the age of forty-seven years, ten years later than the average operative age, recovery from

spontaneous failure is compatible with life for many years in the majority of individuals. We are particularly concerned about its significance because we have recognized permanent cardiac enlargement in some of these individuals, the enlargement being predominantly in chambers proximal to the mitral valve.[12] On the other hand, cardiac enlargement that occurs after surgery is not always reflected in the functional state. Indeed, dyspnea may be relieved by latent right heart failure that becomes obvious if cardiac therapy is withheld. Furthermore, failure occurring months or years after surgery is frequently predominantly right sided. Dyspnea may be absent or a minor complaint although the patient may be obviously short of breath.

This brief review suggests that present day mitral valvotomy carries a higher mortality and morbidity than that which occurs naturally. It is hoped, as with all new surgical procedures, that, with increasing knowledge and better selection of patients, mortality and morbidity will decrease and be justified by the beneficial effects of the operation. It is important, however, that we have unquestionable evidence of surgically induced benefits.

At the very beginning of the present surgical era, it was recognized that a decrease in impediment to blood flow through the mitral orifice would be reflected in a decrease in pressure in the left atrium. Pressure changes at operation obtained at that time were highly variable both in degree and rarely in direction. Discordant changes were seen in systolic and diastolic readings. The degree of atrial distension at the beginning of ventricular contraction could not be assured.[13] It was soon realized that such changes could not be interpreted without knowledge of the pressures on both sides of the obstruction, namely the left atrial systolic pressure, the left ventricular end-diastolic pressure, and also of the cardiac output. The numerous difficulties in obtaining such readings that are reliable are obvious, but such studies are now being carried out, at least in part, in many clinics so that the immediate hemodynamic effects of the surgical procedure will have objective verification. However, even if the atrial-ventricular pressure gradient is decreased at operation without increase in the end-diastolic pressure in the left ventricle and without diminution in cardiac output, persistence of such changes are not guaranteed for several reasons. First, even though the rigid finger may be able to decompress the left atrium, the flow of blood may be inadequate to prevent after a variable time

the elastic properties of the valve from returning the leaflets to their pre-valvotomy position, an occurrence that is readily observed at times at surgery when the finger is withdrawn. So far as we can tell from our pathologic data, calcified leaflets regarded as adequately opened by the surgeon, may be in a position that makes the mitral orifice identical with similar valves that have not been subjected to operation. Secondly, subvalvular stenosis may be unrelieved by surgery. Thirdly, reactivation of rheumatic fever may nullify any potential benefits of operation for it is the viable and pliable mitral valve that may be the seat of fresh Aschoff bodies. Fourthly, mitral regurgitation even of minor degree produced by surgery may nullify beneficial effects of valvotomy. Lastly, changes by surgery in valve size or mobility may be unable to increase left atrial-ventricular flow, the crux of the problem in those with diminished left atrial-ventricular flow.

Most attempts to evaluate permanent effects of the operation on valve function are based upon the technique of right heart catheterization, the results of which are used to infer changes within the left atrium which in turn are attributed to change in valve size and mobility produced by the surgeon. The clinical physiologist is aware that these pressure pulses represent the end result of the interaction of many factors of which impediment to left atrial-ventricular flow is but one. Many acute self-limiting precipitating events can raise these pressures. The reliability of the interpretation of pressure changes will depend first upon the care with which such instances are excluded. Nor are we certain as yet of the time necessary for complete convalescence from such acute events.

Attempts to exclude abnormal pressure findings of myocardial failure from mitral block are based upon the response of these pressures and the cardiac output to digoxin given intravenously.[14] In our experience, the time interval of these studies is far too short to obtain the maximal beneficial effects of digitalis. Moreover, the salutary effects of physical rest itself have been largely ignored. Most important, not all types of myocardial failure respond to digitalis and one of these types is active rheumatic carditis. Sharp drops in cardiac function without obvious cause in individuals with rheumatic heart disease were, in the days before mitral valvotomy, always regarded as suspicious for active rheumatic carditis. Every large clinic has necropsy evidence to support such a diagnosis. Interpretation of pressure changes in such

instances may be very misleading. We have evidence at autopsy of diffuse rheumatic carditis in individuals who were subjected to mitral valvotomy because they had no clinical or laboratory evidence of infection and failed to respond to digitalis. Their only symptom was progressive dyspnea of several months' duration without fever, tachycardia or laboratory evidence of infection.

We are also uncertain of the significance of drops in pressure as the circulation slows or the heart enlarges. It is generally known that pulmonary artery pressure cannot be correlated with cardiac size or cardiac failure, two findings that have been valuable in evaluating prognosis. At this time, we still believe that enlargement of cardiac chambers after surgery outweighs any potential beneficial effects of drops in pulmonary pressure. It may be that transient rises in pulmonary pressure which recede partly as the heart enlarges may be the natural changes that occur as the heart deteriorates. Finally, many reports in the literature indicate that postoperative pulmonary artery pressures (and other numerical figures) may frequently be higher than preoperative pressures of other individuals in the same series. Yet, the first are regarded as indicating marked improvement and the second as requiring surgery. These findings emphasize the lack of correlation even between symptoms or clinical status and hemodynamic events. Indeed, good clinical results are reported in individuals whose pulmonary artery pressure fell, remained unchanged or even rose after operation. We do not understand how improvement can be attributed to mitral valvotomy in those individuals whose pulmonary artery pressure rises after surgery.

Significant obstruction at the mitral valve must be reflected in an increase in pressure proximal to the valve or a decrease in left atrial-ventricular flow or both. A decrease in pressure after mitral valvotomy in chambers proximal to the mitral valve is beneficial only if left atrial-ventricular flow is not simultaneously reduced. For this reason, we have turned our attention at this time primarily to methods that are concerned with left atrial-ventricular flow and the effects upon it of mitral valvotomy and other variables.

We use the biplane stereoscopic apparatus of Chamberlain, timed by the carotid pulse. Simultaneous views in two planes are needed to identify with certainty portions of the cardiovascular silhouette, to visualize opacification in specific regions and to estimate volume. Stereo-

scopy enhances accuracy and is of particular value in differentiating extracardiac densities that may appear to be within the heart on a single angiocardiogram. A mechanical rather than electrical timer is used because many electrical events, such as premature beats or other arrhythmias, may be ineffective in producing mechanical systole of the heart. Timing is necessary in assessing the significance of difference in opacification in serial roentgenograms and in determining the number of cardiac cycles required to fill or empty any portion of the heart. We use 50 milliliters of 70 per cent Urokon injected as rapidly as possible into an exposed antecubital vein. Anterior and lateral projections are used because of greater ease of constancy of reproduction not only in positioning of patient but also in identifying specific regions without overlapping. Roentgenograms are obtained every 0.7 second for at least 21 seconds. Subsequent roentgenograms are obtained at variable intervals depending upon the clinical evaluation of heart function and the radiographic determination of heart size. The tube is shifted at the time of exposure of alternate films to allow for stereoscopic pairs. The roentgenograms are analyzed for time of appearance and disappearance of opacity of the superior vena cava, right heart, pulmonary artery, left atrium, left ventricle and aorta. The degree of opacification is measured and corroborated by a densitometer. The results are plotted as density against time. Details of this study will be published elsewhere.[15]

In the normal individual, the time from the initial appearance of dye within the right atrium to the appearance of dye in the ascending aorta, which we call the intracardiac circulation time that represents the shortest time required for dye in detectable concentration to traverse the heart, is not greater than 6.3 seconds and always exceeds the right heart time. The right heart time, pulmonary artery time and left atrial times approximate each other and are each 5.6 seconds or less. Opacification of the pulmonary artery disappears before the ascending aorta is visualized. Left atrial density is not great and is poorly delimited. Indeed, dye opacifies clearly the pulmonary veins adjacent to the left atrium more than the left atrium itself and fuses quickly with the left ventricular density. The left ventricle is opacified usually in less than 0.7 second and never longer than 1.4 seconds after initial left atrial opacification.

These findings are in marked contrast to those in individuals with

mitral stenosis. In the absence of heart failure or marked cardiomegaly, the intracardiac circulation time may be prolonged but does not exceed 9.1 seconds. The right heart time is usually less than the intracardiac circulation time and rarely may equal it. The pulmonary artery time may be much longer than the right heart time and the pulmonary artery may remain visualized after the appearance of the ascending aorta. The left atrial time is 6.3 seconds or longer and almost always exceeds both the pulmonary artery and intracardiac circulation times. The left atrium is increased in size and its borders are sharply defined. Visualization of the left ventricle occurs within 0.7 second or may be delayed to as long as 2.8 seconds after initial left atrial opacification. Opacification of the left ventricle is sparse compared to that of the left atrium. However, the difference between left atrial and left ventricular density depends not only upon time but also upon the phase in the cardiac cycle that the roentgenogram is exposed. It is possible to have a series of roentgenograms without opacifying the ventricle once in diastole. Conversely, ventricular diastole may be visualized several times. Also, left ventricular opacification may be readily apparent in one projection and impossible to recognize in another. Finally, stereoscopy allows for recognition of dye within the pulmonary vasculature which appears to be within the left ventricle in a single angiocardiogram.

The finding in mitral stenosis of a left atrial time longer than that of the intracardiac circulation time and of the pulmonary artery time, which, in turn, in the absence of right heart failure, is usually longer than the right heart time, is visual evidence supporting the concept of a block at the mitral valve.

The angiocardiographic pattern of mitral stenosis which we have just described is, however, not diagnostic. A prolonged left atrial time and sparse opacification of the left ventricle may be seen in mitral stenosis with regurgitation or even in pure mitral regurgitation as diagnosed by the surgeon. A giant left atrium with marked regurgitation and minimal stenosis diagnosed at operation likewise gives the picture of an intensely opacified left atrium with delay and sometimes complete non-opacification of the left ventricle. No individual with clinically diagnosed pure mitral regurgitation was subjected to operation. Individuals so diagnosed clinically with but slightly enlarged left atria showed homogeneous opacification of the left atrium and left

ventricle. Otherwise, no relationship could be determined between left atrial time and left atrial size or between left atrial and ventricular opacification to differentiate the diagnosis of "pure" mitral stenosis from mitral stenosis and regurgitation as made at surgery or to predict the degree of mitral stenosis as reported by the surgeon. However, a lifting apical thrust was found in all individuals with large atria.

Indeed, the entire angiocardiographic pattern of mitral stenosis may occur in the complete absence of organic mitral valve disease. Apparently, non-valvular enlargement of the left atrium may in itself impede left atrial-ventricular flow. We have seen such findings in an individual with primary pulmonary artery disease, proved at autopsy.

However, the possibility of occult left ventricular failure in this instance could not be excluded, because it is easy to show that left ventricular failure of any cause, be it aortic valve disease, systemic hypertension or coronary artery disease, may produce the left atrial pattern of mitral valve disease. The distinction between the two is, however, easy, because of the thickness of the left ventricular wall, increase in degree and duration of left ventricular opacification and with aortic disease frequent eccentric opacification of the aortic orifice. Additional mitral stenosis, however, if present, would be difficult if not impossible to detect by angiocardiography.

These findings visualize the clinical physiologist's pressure findings in left ventricular failure but frequently precede them because such changes are seen before clinical failure is recognizable.

Indeed, in our experience, the angiocardiographic pattern of mitral stenosis precedes abnormal pulmonary artery or pulmonary artery wedge pressures. It is for this reason that we believe that slowing of the circulation may occur before rises in pressure or may persist after pressure falls.

It is clear that angiocardiography, like other techniques, is not specific for mitral stenosis and that impediment of left atrial emptying, as determined by this technique, can occur in those with mitral valve disease, regardless of whether stenosis, regurgitation or both are present and can occur even in the absence of organic valvular disease.

Although the angiocardiographic pattern is not specific for mitral stenosis, it was hoped that mitral valvotomy would abolish or decrease the impediment to blood flow in individuals who did have mitral stenosis as visualized by this technique. Our studies, however, indicate

that present day surgery for mitral stenosis cannot abolish impediment of left atrial-ventricular flow as visualized by this technique, nor are there any characteristic angiocardiographic features, other than absence of the left auricle or presence of thoracic surgical incisions, that can differentiate those who have had mitral valvotomy from those who have not. Any change is a purely qualitative one. We have already indicated, however, that impediment of left atrial-ventricular flow of similar magnitude may be due to many factors other than mitral stenosis. It is important that qualitative changes in left atrial-ventricular flow due to treatment of these factors should not be interpreted as due to surgical treatment of mitral stenosis.

The significance of medical therapy in reducing impediment of left atrial-ventricular flow can be shown in some individuals who have already had their mitral stenosis reduced by surgery. An intensified medical regimen in individuals with congestive heart failure or even with dyspnea alone may shorten all circulation times, disproportionately shorten the left atrial time and increase degree of left ventricular opacification.

This last change with medical treatment indicates that the pattern of mitral stenosis in the same individual is more often seen in failure than in compensation, evidence of the importance of myocardial function in left atrial-ventricular flow. Changes of this sort are most quickly made by mercurials and increased digitalis and may be accomplished by digitalis alone or even by bed rest without other change in cardiac therapy. But, maximal changes take time and, in our experience, at least six weeks and possibly longer. Such changes can, at times, be demonstrated even in the absence of clinical evidence of heart failure and with an apparently adequately controlled ventricular response to digitalis in individuals with atrial fibrillation.

Mitral valvotomy, therefore, not only cannot abolish left atrial-ventricular block but also cannot be evaluated unless all other medication is kept constant. Indeed, the problem is even more difficult because some disabilities respond only to rest and time, such as carditis, pulmonary infarction and even infection. It is for this reason that we have stressed the need in interpreting the effect of mitral valvotomy for static disability maximally controlled by medical therapy.

Because of the present day attitude, it is difficult to obtain adequate controls. But, whenever the individual appeared stabilized clinically for

at least one month, we have, as yet, not observed any improvement in left atrial-ventricular flow comparable to that seen with medical treatment.

Of thirty individuals who regard themselves as improved after operation, nine had atrial fibrillation and large hearts estimated above 600 cc. before operation. All have persistent and markedly prolonged left atrial and pulmonary artery times. Three of these greatly exceed their preoperative times. Pressure studies in apparently comparable individuals have shown variable and usually slight changes. Even if marked drops in pulmonary artery pressures are obtained, it is difficult to interpret their significance because it is in these larger hearts that angiocardiography, in the immediate postoperative period, may demonstrate a significant increase in size of the pulmonary artery and of the right atrium and right ventricle with slowing of the intracardiac circulation, and with decreased opacification and, at times non-opacification of the left ventricle. These findings persist. They may not be reflected in recognizable manifestations but this type of individual represents one we have previously described who may develop clinical right heart failure after operation. These patients, although worse, may genuinely feel better because troublesome dyspnea has been replaced by right heart failure, the clinical manifestations of which, if present, may be controlled by mercurial injections. Indeed, right heart failure may be present in individuals who deny ever having had dyspnea. Right heart failure may not be recognized on conventional films. Because of the apparent decrease in pulmonary vascular markings, the radiologist might interpret the increased heart size as due to increased left ventricular filling. Yet, angiocardiography reveals marked slowing of the circulation through the right heart and pulmonary artery, and no dye within the left ventricle. In this type of failure, the jugular veins are prominent and fill rapidly from below. An elevated venous pressure may be demonstrated by abdominal compression.

Two with only slightly enlarged hearts developed permanent atrial fibrillation and more prolonged left atrial and pulmonary artery times after surgery.

Three had atrial fibrillation with slightly enlarged hearts. Two of these had persistently long left atrial and pulmonary artery times. The other, who appears to us considerably better clinically, has a prolonged left atrial time but a normal pulmonary artery time.

Sixteen had hearts of normal or slightly increased size with sinus rhythm. Of these, six have persistent markedly prolonged left atrial and pulmonary artery times. Ten have persistent slightly prolonged left atrial and pulmonary artery times. We could not distinguish these last two groups on clinical grounds. One patient who stated she had one block dyspnea for over six months, uninfluenced by digitalis, had a pliable mitral valve that was easily adequately opened and now leads an entirely normal life. However, she has a persistent right heart time of 11.9 seconds, a pulmonary artery time of 12.6 seconds and a left atrial time of 14 seconds. A retrospective history raised the possibility of preoperative psychogenic dyspnea engendered by the present surgical climate. She had forced her doctor to consent to operation and we agreed because of unequivocal findings of pulmonary hypertension. She was catheterized four days ago, ten months after operation. Her right ventricular pressure is 50/8 mm. Hg and her pulmonary artery pressure is 52/30 mm. Hg. From these studies, it appears that, in contrast to myocardial function that has an obviously potent effect on left atrial-ventricular flow, mitral valvotomy has none or one too feeble to be detected by our technique. Mitral valvotomy, on the other hand, may permanently decrease left atrial-ventricular flow in individuals with a large heart and in individuals who develop permanent atrial fibrillation. In the individual with mitral stenosis and a small heart, near normal circulation times may be maintained by a disproportionately high pressure proximal to the obstruction. The main purpose of mitral valvotomy is to permit such flow to continue at more nearly normal pressures. However, near normal times in individuals with mitral stenosis may be present with normal or near normal right ventricular, pulmonary artery and pulmonary artery wedge pressures.

For these reasons, we believe, at the present time, that the first responsibility of a physician when confronted with an individual with mitral stenosis, is to determine if symptoms are due to self-limiting or medically treatable events. If in doubt, there is hardly ever any urgency to operate. Our angiocardiographic studies suggest, on the basis of comparison with conventional films, some of which were taken as long as twenty years ago, that mitral block, in the absence of precipitating events, changes so slowly, if at all, that for practical purposes it may be regarded as stationary for years. Even repeated attacks of pulmonary edema are compatible with long intervals of freedom from

symptoms and need for cardiac therapy. Thus one individual had pulmonary edema twenty years ago during her first pregnancy, eight years ago during her fifth pregnancy and one month before the present study while rushing home from a party. Between attacks she had no symptoms and no cardiac treatment. Her intracardiac circulation time is 7.7 seconds, her right heart time 4.9 seconds, her pulmonary artery time 6.3 seconds, her left atrial time 7 seconds. Her right ventricular pressure was 45/3 mm. Hg and her pulmonary artery pressure 45/22 mm. Hg.

The second responsibility of the physician is to determine if simple precautionary measures do not produce a comfortable and satisfactory life for the individual's age, social and economic status. If not, and pulmonary artery pressures are high, mitral valvotomy may be advised but not urged.

Mitral valvotomy should never be urged because it is only those with normal or slightly enlarged hearts and no roentgen-ray evidence of calcification of the mitral valve who possibly may be benefited. These individuals have the best natural prognosis so far as the ultimate development of congestive failure is concerned. It is rare for them to die before the sixth decade. Surgery, on the other hand, has even in this group an immediate mortality of at least 4 per cent, and at least 2 per cent chance of embolism in those who are fibrillating and survive operation and a 5 to 10 per cent chance of the production of permanent atrial fibrillation. Moreover, the diagnosis may be wrong. The operation never destroys mitral block completely and indeed is rarely effective in individuals with densely fibrotic and motionless valves. Finally, the duration of benefit, if any, is as yet unknown and involves the concept of restenosis.

Mitral block is never abolished by present day surgery. The clinical concept of restenosis is, therefore meaningless. The degree of mitral block may increase with time after surgery but the cause for this increase is conjectural. We do know from pathologic studies that the incisural surfaces of calcified, fibrotic and motionless valves do not reseal up to five years. But, from the obstructive standpoint, it makes no or very little difference if these surfaces reseal or remain permanently separated from one another by a distance equivalent to the thickness of the surgeon's knife or gape widely after finger fracture.

Restenosis has been used by many as synonomous with reappear-

ance of symptoms. Reappearance of symptoms after operation may appear for the same reasons that they occur before operation. Active carditis, arrhythmias, infection, pulmonary infarction, and excessive physical strain are common causes. Symptoms will depend upon the interaction of these precipitating events and the cardiac reserve. It is hoped that the earlier in the life history of rheumatic fever mitral valvotomy is done and the smaller the heart, the longer will the patient remain free of symptoms after operation because this is indeed true for the individual without surgery.

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